

Anti-MLH3 antibody ab4834

1 References [画像数 1](#)

製品の概要

製品名	Anti-MLH3 antibody
製品の詳細	Goat polyclonal to MLH3
由来種	Goat
アプリケーション	適用あり: ICC/IF
種交差性	交差種: Human
免疫原	Synthetic peptide: RQSLQQSMPPCEPP , corresponding to C terminal amino acids 1416-1429 of Human MLH3.

 [Run BLAST with](#)

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特記事項	<p>Gene Ontology terms - satellite DNA binding; ATP binding; molecular_function unknown; mismatch repair; biological_process unknown; nucleus; cellular_component unknown</p> <p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As</p>
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製品の特性

製品の状態	Liquid
保存方法	Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
バッファー	pH: 7.30 Preservative: 0.02% Sodium azide Constituents: Tris buffered saline, 0.5% BSA
精製度	Immunogen affinity purified
特記事項 (精製)	Purified from goat serum by ammonium sulphate precipitation followed by antigen affinity chromatography using the immunizing peptide.
ポリ/モノ	ポリクローナル

アプリケーション

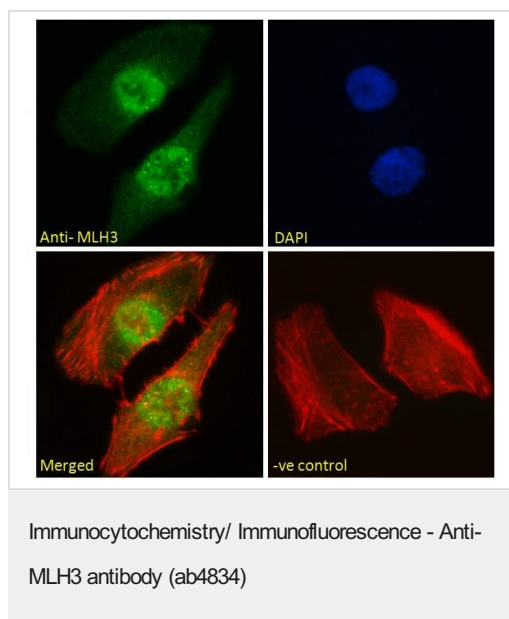
The Abpromise guarantee Abpromise保証は、 次のテスト済みアプリケーションにおけるab4834の使用に適用されます
アプリケーションノートには、推奨の開始希釈率がありますが、適切な希釈率につきましてはご検討ください。

アプリケーション	Abreviews	特記事項
ICC/IF		Use a concentration of 10 µg/ml.

ターゲット情報

機能	Probably involved in the repair of mismatches in DNA.
組織特異性	Ubiquitous.
関連疾患	<p>Defects in MLH3 are the cause of hereditary non-polyposis colorectal cancer type 7 (HNPCC7) [MIM:614385]. Mutations in more than one gene locus can be involved alone or in combination in the production of the HNPCC phenotype (also called Lynch syndrome). Most families with clinically recognized HNPCC have mutations in either MLH1 or MSH2 genes. HNPCC is an autosomal, dominantly inherited disease associated with marked increase in cancer susceptibility. It is characterized by a familial predisposition to early onset colorectal carcinoma (CRC) and extra-colonic cancers of the gastrointestinal, urological and female reproductive tracts. HNPCC is reported to be the most common form of inherited colorectal cancer in the Western world, and accounts for 15% of all colon cancers. Cancers in HNPCC originate within benign neoplastic polyps termed adenomas. Clinically, HNPCC is often divided into two subgroups. Type I: hereditary predisposition to colorectal cancer, a young age of onset, and carcinoma observed in the proximal colon. Type II: patients have an increased risk for cancers in certain tissues such as the uterus, ovary, breast, stomach, small intestine, skin, and larynx in addition to the colon. Diagnosis of classical HNPCC is based on the Amsterdam criteria: 3 or more relatives affected by colorectal cancer, one a first degree relative of the other two; 2 or more generation affected; 1 or more colorectal cancers presenting before 50 years of age; exclusion of hereditary polyposis syndromes. The term 'suspected HNPCC' or 'incomplete HNPCC' can be used to describe families who do not or only partially fulfill the Amsterdam criteria, but in whom a genetic basis for colon cancer is strongly suspected.</p> <p>Defects in MLH3 are a cause of colorectal cancer (CRC) [MIM:114500].</p>
配列類似性	Belongs to the DNA mismatch repair mutL/hexB family.
細胞内局在	Nucleus.

画像



Immunofluorescence analysis of paraformaldehyde fixed HeLa cells, permeabilized with 0.15% Triton. Primary incubation with ab4834 for 1hr (10 µg/ml) followed by Alexa Fluor 488 secondary antibody (2 µg/ml), showing nuclear staining. Actin filaments were stained with phalloidin (red) and the nuclear stain is DAPI (blue). Negative control: Unimmunized goat IgG (10 µg/ml) followed by Alexa Fluor 488 secondary antibody (2 µg/ml).

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